```
=> d his

     (FILE 'HOME' ENTERED AT 17:31:41 ON 08 JAN 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:33:38 ON 08 JAN
     2004
L1        681133 S GROWTH(W)FACTOR OR NGF OR NT-3 OR NEUROTROPHIN
L2         14928 S (DNA OR NUCLEIC(W)ACID OR POLYNUCLEOTIDE OR CDNA OR TRANSGENE
L3       3001424 S (NEURONAL(W)ATROPHY OR BRAIN OR ALZHEIMER OR PARKINSON OR AD
L4           449 S L2(S)L3
L5           247 S L2(8A)L3
L6           185 S L2(5A)L3
L7           142 DUP REM L6 (43 DUPLICATES REMOVED)
L8        205336 S (AMELIORAT? OR INHIBIT? OR DECREAS? OR TREAT? OR ABOLISH?)(8A
L9            48 S L2(S)L8
L10           38 DUP REM L9 (10 DUPLICATES REMOVED)

=> d au ti so 1-38 l10

L10  ANSWER 1 OF 38  CAPLUS  COPYRIGHT 2004 ACS on STN DUPLICATE 1
IN   Akhtar, Saghir; McSwiggen, James
TI   Enzymatic nucleic acid treatment of diseases or conditions related to
     levels of epidermal growth factor receptors
SO   U.S. Pat. Appl. Publ., 199 pp., Cont.-in-part of U.S. Ser. No. 401,063.
     CODEN: USXXCO

L10  ANSWER 2 OF 38  SCISEARCH  COPYRIGHT 2004 THOMSON ISI on STN
AU   Wu K (Reprint); Klein R L; Meyers C A; King M A; Hughes J A; Millard W J;
     Meyer E M
TI   Long-term neuronal effects and disposition of ectopic preproNGF gene
     transfer into the rat septum
SO   HUMAN GENE THERAPY, (OCT 2003) Vol. 14, No. 15, pp. 1463-1472.
     Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY
     10538 USA.
     ISSN: 1043-0342.

L10  ANSWER 3 OF 38  SCISEARCH  COPYRIGHT 2004 THOMSON ISI on STN
AU   Ha X Q; Yuan B; Li Y M; Lao M F; Wu Z Z (Reprint)
TI   Gene therapy for pathological scar with hepatocyte growth factor mediated
     by recombinant adenovirus vector
SO   SCIENCE IN CHINA SERIES C-LIFE SCIENCES, (JUN 2003) Vol. 46, No. 3, pp.
     320-+.
     Publisher: SCIENCE CHINA PRESS, 16 DONGHUANGCHENGGEN NORTH ST, BEIJING
     100717, PEOPLES R CHINA.
     ISSN: 1006-9305.

L10  ANSWER 4 OF 38     MEDLINE on STN                    DUPLICATE 2
AU   Donnerer Josef
TI   Regeneration of primary sensory neurons.
SO   PHARMACOLOGY, (2003 Apr) 67 (4) 169-81.  Ref: 81
     Journal code: 0152016. ISSN: 0031-7012.

L10  ANSWER 5 OF 38  CAPLUS  COPYRIGHT 2004 ACS on STN
IN   Bringmann, Peter W.; Faulds, Daryl; Mitrovic, Bransilava; Srinivasan,
     Subha
TI   cDNA and protein sequences of human fibroblast growth factors, FGF-20 and
     FGF-23, and their used in treatment of diseases
SO   PCT Int. Appl., 55 pp.
     CODEN: PIXXD2

L10  ANSWER 6 OF 38  CAPLUS  COPYRIGHT 2004 ACS on STN
IN   Mao, Yumin; Xie, Yi
TI   Human 10.67-kDa vascular endothelial growth factor 165 receptor like
```

protein and its cDNA and therapeutic use
SO Faming Zhuanli Shenqing Gongkai Shuomingshu, 34 pp.
CODEN: CNXXEV

L10 ANSWER 7 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Kee H J; Koh J T; Kim M Y; Ahn K Y; Kim J K; Bae C S; Park S S; Kim K K (Reprint)
TI Expression of brain-specific angiogenesis inhibitor 2 (BAI2) in normal and ischemic brain: Involvement of BAI2 in the ischemia-induced brain angiogenesis
SO JOURNAL OF CEREBRAL BLOOD FLOW AND METABOLISM, (SEP 2002) Vol. 22, No. 9, pp. 1054-1067.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0271-678X.

L10 ANSWER 8 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Cao Y J; Shibata T; Rainov N G (Reprint)
TI Liposome-mediated transfer of the bcl-2 gene results in neuroprotection after in vivo transient focal cerebral ischemia in an animal model
SO GENE THERAPY, (MAR 2002) Vol. 9, No. 6, pp. 415-419.
Publisher: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND.
ISSN: 0969-7128.

L10 ANSWER 9 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Zhang Y; Zhu C N; Pardridge W M (Reprint)
TI Antisense gene therapy of brain cancer with an artificial virus gene delivery system
SO MOLECULAR THERAPY, (JUL 2002) Vol. 6, No. 1, pp. 67-72.
Publisher: ACADEMIC PRESS INC ELSEVIER SCIENCE, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA.
ISSN: 1525-0016.

L10 ANSWER 10 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Su, Eric Wen; Na, Songqing
TI Human fibroblast growth factor-20 nucleic acids and polypeptides
SO PCT Int. Appl., 137 pp.
CODEN: PIXXD2

L10 ANSWER 11 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Vernet, Corine A. M.; Fernandes, Elma; Shimkets, Richard A.; Herrmann, John L.; Majumder, Kumud; MacDougall, John; Mishra, Vishnu; Mezes, Peter S.; Rastelli, Luca
TI Human FCTR proteins related to growth factors and cDNAs encoding them and their use in drug screening and therapy
SO PCT Int. Appl., 215 pp.
CODEN: PIXXD2

L10 ANSWER 12 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Thomason, Arlen Read; Liu, Benxian
TI Mouse and human homologs of fibroblast growth factor with possible therapeutic use and cDNAs encoding them
SO PCT Int. Appl., 138 pp.
CODEN: PIXXD2

L10 ANSWER 13 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Ruben, Steven M.; Jimenez, Pablo; Duan, D. Roxannes; Rampy, Mark A.; Mendrick, Donna; Zhang, Jun; Ni, Jian; Moore, Paul A.; Coleman, Timothy A.; Gruber, Joachim R.; Dillon, Patrick J.; Gentz, Reiner L.
TI Cloning, cDNA sequence and biological activities of human keratinocyte growth factor-2
SO PCT Int. Appl., 592 pp.
CODEN: PIXXD2

L10 ANSWER 14 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Shinoda M (Reprint); Hidaka M; Lindqvist E; Soderstrom S; Matsumae M; Oi S; Sato O; Tsugane R; Ebendal T; Olson L
TI NGF, NT-3 and Trk C mRNAs, but not TrkA mRNA, are upregulated in the paraventricular structures in experimental hydrocephalus
SO CHILDS NERVOUS SYSTEM, (DEC 2001) Vol. 17, No. 12, pp. 704-712.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010 USA.
ISSN: 0256-7040.

L10 ANSWER 15 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AU Ay, I.; Sugimori, H.; Finklestein, S. P.
TI Intravenous basic fibroblast growth factor (bFGF) decreases DNA fragmentation and prevents downregulation of Bcl-2 expression in the ischemic brain following middle cerebral artery occlusion in rats
SO Molecular Brain Research (2001), 87(1), 71-80
CODEN: MBREE4; ISSN: 0169-328X

L10 ANSWER 16 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Kavanaugh, Michael; Shyamala, Venkatakrishna; Reinhard, Christoph
TI Protein and cDNA sequences encoding human fibroblast growth factor (FGF-X), and uses thereof in the diagnosis and treatment of pulmonary diseases and disorders associated with neuronal cell degeneration
SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2

L10 ANSWER 17 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Geerts, Hugo Alfonso; Masure, Stefan Leo Jozef; Meert, Theo Frans; Cik, Miroslav; Ver Donck, Luc August Laurentius
TI Human neurotrophic growth factor enovin and its encoding nucleic acids and biological activities
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2

L10 ANSWER 18 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Printz M A (Reprint); Gonzalez A M; Cunningham M; Gu D L; Ong M; Pierce G F; Aukerman S L
TI Fibroblast growth factor 2-retargeted adenoviral vectors exhibit a modified biolocalization pattern and display reduced toxicity relative to native adenoviral vectors
SO HUMAN GENE THERAPY, (1 JAN 2000) Vol. 11, No. 1, pp. 191-204.
Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY 10538.
ISSN: 1043-0342.

L10 ANSWER 19 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Johnson, Eugene M.; Milbrandt, Jeffrey D.; Kotzbauer, Paul T.; Lampe, Patricia A.; Klein, Robert; Desauvage, Fred
TI Nucleic acids encoding mammalian persephin and neurturin growth factors
SO PCT Int. Appl., 222 pp.
CODEN: PIXXD2

L10 ANSWER 20 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Gwag B J; Canzoniero L M T; Sensi S L; Demaro J A; Koh J Y; Goldberg M P; Jacquin M; Choi D W (Reprint)
TI Calcium ionophores can induce either apoptosis or necrosis in cultured cortical neurons
SO NEUROSCIENCE, (APR 1999) Vol. 90, No. 4, pp. 1339-1348.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0306-4522.

L10 ANSWER 21 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Patel S R; Lee L Y; Mack C A; Polce D R; ElSawy T; Hackett N R; Ilercil A;

Jones E C; Hahn R T; Isom O W; Rosengart T K; Crystal R G (Reprint)
TI Safety of direct myocardial administration of an adenovirus vector encoding vascular endothelial growth factor 121
SO HUMAN GENE THERAPY, (20 MAY 1999) Vol. 10, No. 8, pp. 1331-1348.
Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY 10538.
ISSN: 1043-0342.

L10 ANSWER 22 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Zou L L; Huang L; Hayes R L; Black C; Qiu Y H; PerezPolo J R; Le W; Clifton G L; Yang K (Reprint)
TI Liposome-mediated NGF gene transfection following neuronal injury: potential therapeutic applications
SO GENE THERAPY, (JUN 1999) Vol. 6, No. 6, pp. 994-1005.
Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0969-7128.

L10 ANSWER 23 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Im S A; GomezManzano C; Fueyo J; Liu T J; Ke L D; Kim J S; Lee H Y; Steck P A; Kyritsis A P; Yung W K A (Reprint)
TI Antiangiogenesis treatment for gliomas: Transfer of antisense vascular endothelial growth factor inhibits tumor growth in vivo
SO CANCER RESEARCH, (15 FEB 1999) Vol. 59, No. 4, pp. 895-900.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202.
ISSN: 0008-5472.

L10 ANSWER 24 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Mandel R J (Reprint); Gage F H; Clevenger D G; Spratt S K; Snyder R O; Leff S E
TI Nerve growth factor expressed in the medial septum following in vivo gene delivery using a recombinant adeno-associated viral vector protects cholinergic neurons from fimbria-fornix lesion-induced degeneration
SO EXPERIMENTAL NEUROLOGY, (JAN 1999) Vol. 155, No. 1, pp. 59-64.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0014-4886.

L10 ANSWER 25 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Nathans, Jeremy; Smallwood, Philip M.
TI Human fibroblast growth factor homologous factors and their cDNAs and diagnostic and therapeutic applications
SO PCT Int. Appl., 95 pp.
CODEN: PIXXD2

L10 ANSWER 26 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Wortwein G; Yu J; ToliverKinsky T; PerezPolo J R (Reprint)
TI Responses of young and aged rat CNS to partial cholinergic immunolesions and NGF treatment
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1 MAY 1998) Vol. 52, No. 3, pp. 322-333.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0360-4012.

L10 ANSWER 27 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Worley, Paul; Tsui, Cynthia
TI A neuronal cell activity regulated pentraxin as a neuronal cell growth factor and a cDNA encoding it
SO PCT Int. Appl., 104 pp.
CODEN: PIXXD2

L10 ANSWER 28 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU GalveRoperh I; Malpartida J M; Haro A; Brachet P; DiazLaviada I (Reprint)

TI Regulation of nerve growth factor secretion and mRNA expression by bacterial lipopolysaccharide in primary cultures of rat astrocytes
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1 SEP 1997) Vol. 49, No. 5, pp. 569-575.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0360-4012.

L10 ANSWER 29 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AU Tao, Y.; Black, I. B.; Dicicco-Bloom, E.
TI Neurogenesis in neonatal rat brain is regulated by peripheral injection of basic fibroblast growth factor (bFGF)
SO Journal of Comparative Neurology (1996), 376(4), 653-663
CODEN: JCNEAM; ISSN: 0021-9967

L10 ANSWER 30 OF 38 MEDLINE on STN DUPLICATE 3
AU Iwamoto Y; Yang K; Clifton G L; Hayes R L
TI Liposome-mediated BDNF cDNA transfer in intact and injured rat brain.
SO NEUROREPORT, (1996 Jan 31) 7 (2) 609-12.
Journal code: 9100935. ISSN: 0959-4965.

L10 ANSWER 31 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AU Nakaya, Naoki; Nishibori, Masahiro; Kawabata, Masahiro; Saeki, Kiyomi
TI Cloning of a serine proteinase inhibitor from bovine brain: expression in the brain and characterization of its target proteinases
SO Molecular Brain Research (1996), 42(2), 293-300
CODEN: MBREE4; ISSN: 0169-328X

L10 ANSWER 32 OF 38 MEDLINE on STN DUPLICATE 4
AU Muhlhauser J; Merrill M J; Pili R; Maeda H; Bacic M; Bewig B; Passaniti A; Edwards N A; Crystal R G; Capogrossi M C
TI VEGF165 expressed by a replication-deficient recombinant adenovirus vector induces angiogenesis in vivo.
SO CIRCULATION RESEARCH, (1995 Dec) 77 (6) 1077-86.
Journal code: 0047103. ISSN: 0009-7330.

L10 ANSWER 33 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU DIMBERG Y (Reprint)
TI NEUROCHEMICAL EFFECTS OF A 20 KHZ MAGNETIC-FIELD ON THE CENTRAL-NERVOUS-SYSTEM IN PRENATALLY EXPOSED MICE
SO BIOELECTROMAGNETICS, (1995) Vol. 16, No. 4, pp. 263-267.
ISSN: 0197-8462.

L10 ANSWER 34 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU DERCOLE A J (Reprint); DAI Z H; XING Y Z; BONEY C; WILKIE M B; LAUDER J M; HAN V K M; CLEMMONS D R
TI BRAIN GROWTH-RETARDATION DUE TO THE EXPRESSION OF HUMAN INSULIN-LIKE GROWTH-FACTOR BINDING PROTEIN-1 IN TRANSGENIC MICE - AN IN-VIVO MODEL FOR THE ANALYSIS OF IGF FUNCTION IN THE BRAIN
SO DEVELOPMENTAL BRAIN RESEARCH, (14 OCT 1994) Vol. 82, No. 1-2, pp. 213-222.
ISSN: 0165-3806.

L10 ANSWER 35 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU DECKWERTH T L; JOHNSON E M (Reprint)
TI TEMPORAL ANALYSIS OF EVENTS ASSOCIATED WITH PROGRAMMED CELL-DEATH (APOPTOSIS) OF SYMPATHETIC NEURONS DEPRIVED OF NERVE GROWTH-FACTOR
SO JOURNAL OF CELL BIOLOGY, (DEC 1993) Vol. 123, No. 5, pp. 1207-1222.
ISSN: 0021-9525.

L10 ANSWER 36 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU LOWE W L (Reprint); MEYER T; KARPEN C W; LORENTZEN L R
TI REGULATION OF INSULIN-LIKE GROWTH FACTOR-I PRODUCTION IN RAT C6 GLIOMA-CELLS - POSSIBLE ROLE AS AN AUTOCRINE PARACRINE GROWTH-FACTOR
SO ENDOCRINOLOGY, (MAY 1992) Vol. 130, No. 5, pp. 2683-2691.

ISSN: 0013-7227.

L10 ANSWER 37 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU TORP S H (Reprint); HELSETH E; RYAN L; STOLAN S; DALEN A; UNSGAARD G
TI EXPRESSION OF THE EPIDERMAL GROWTH-FACTOR RECEPTOR GENE IN HUMAN BRAIN METASTASES
SO APMIS, (AUG 1992) Vol. 100, No. 8, pp. 713-719.
ISSN: 0903-4641.

L10 ANSWER 38 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
IN Hohn, Andreas; Leibrock, Joachim; Bailey, Karen; Barde, Yves Alain; Thoenen, Hans F. E.; Maisonpierre, Peter C.; Furth, Mark E.; Lindsay, Ronald M.; Yancopoulos, George
TI Cloning and expression of neurotrophin-3 (NT-3) cDNA and use of NT-3 for diagnosis and treatment of neurologic disorders
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2

=> d bib ab 19-38 l10

L10 ANSWER 19 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:216935 CAPLUS
DN 130:247452
TI Nucleic acids encoding mammalian persephin and neurturin growth factors
IN Johnson, Eugene M.; Milbrandt, Jeffrey D.; Kotzbauer, Paul T.; Lampe, Patricia A.; Klein, Robert; Desauvage, Fred
PA Washington University, USA
SO PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9914235	A1	19990325	WO 1998-US19163	19980915
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6222022	B1	20010424	US 1997-931858	19970916
	AU 9894838	A1	19990405	AU 1998-94838	19980915
	AU 758011	B2	20030313		
	EP 1009768	A1	20000621	EP 1998-948217	19980915
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001516764	T2	20011002	JP 2000-511783	19980915
	NZ 502090	A	20021025	NZ 1998-502090	19980915
PRAI	US 1997-931858	A1	19970916		
	US 1996-615944	B2	19960314		
	WO 1997-US3461	A2	19970314		
	US 1997-881172	B2	19970623		
	WO 1998-US19163	W	19980915		

AB Two novel growth factors, neurturin and persephin, which belongs to the glial-derived neurotrophic factor (GDNF)/neurturin/persephin family of growth factors, are disclosed. The human, mouse, and rat amino acid sequences were identified. Human, mouse and rat persephin genomic DNA sequences were cloned and sequenced and the resp. cDNA sequences identified. In addn., methods for treating degenerative conditions using persephin, methods for detecting persephin gene alterations, and methods

for detecting and monitoring patient levels of persephin are provided. Methods for identifying addnl. members of the persephin-neurturin-GDNF family of growth factors are also provided.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 20 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1999:289315 SCISEARCH
GA The Genuine Article (R) Number: 184UE
TI Calcium ionophores can induce either apoptosis or necrosis in cultured cortical neurons
AU Gwag B J; Canzoniero L M T; Sensi S L; Demaro J A; Koh J Y; Goldberg M P; Jacquin M; Choi D W (Reprint)
CS AJOU UNIV, SCH MED, DEPT PHARMACOL, SUWON 442749, KYUNGKIDO, SOUTH KOREA (Reprint); AJOU UNIV, SCH MED, DEPT PHARMACOL, SUWON 442749, KYUNGKIDO, SOUTH KOREA; WASHINGTON UNIV, SCH MED, DEPT NEUROL, ST LOUIS, MO 63110; WASHINGTON UNIV, SCH MED, CTR STUDY NERVOUS SYST INJURY, ST LOUIS, MO 63110; UNIV ULSAN, SCH MED, DEPT NEUROL, SEOUL, SOUTH KOREA
CYA SOUTH KOREA; USA
SO NEUROSCIENCE, (APR 1999) Vol. 90, No. 4, pp. 1339-1348.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0306-4522.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 79
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Cultured cortical neurons exposed for 24 h to low concentrations of the Ca2+ ionophores, ionomycin (250 nM) or A-23187 (100 nM), underwent apoptosis, accompanied by early degeneration of neurites, cell body shrinkage, chromatin condensation and internucleosomal DNA fragmentation. This death could be blocked by protein synthesis **inhibitors**, as well as by the growth factors **brain**-derived neurotrophic factor or insulin-like growth factor I. If the ionomycin concentration was increased to 1-3 mu M, then neurons underwent necrosis, accompanied by early cell body swelling without **DNA** laddering, or sensitivity to cycloheximide or **growth factors**. Calcium imaging with Fura-2 suggested a possible basis for the differential effects of low and high concentrations of ionomycin. At low concentrations, ionomycin induced greater increases in intracellular Ca2+ concentration in neurites than in neuronal cell bodies, whereas at high concentrations, ionomycin produced large increases in intracellular Ca2+ concentration in both neurites and cell bodies.

We hypothesize that the ability of low concentrations of Ca2+ ionophores to raise intracellular Ca2+ concentration preferentially in neurites caused early neurite degeneration, leading to loss of growth factor availability to the cell body and consequent apoptosis, whereas high concentrations of ionophores produced global cellular Ca2+ overload and consequent necrosis. (C) 1999 IBRO. Published by Elsevier Science Ltd.

L10 ANSWER 21 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1999:413111 SCISEARCH
GA The Genuine Article (R) Number: 199QQ
TI Safety of direct myocardial administration of an adenovirus vector encoding vascular endothelial growth factor 121
AU Patel S R; Lee L Y; Mack C A; Polce D R; ElSawy T; Hackett N R; Ilercil A; Jones E C; Hahn R T; Isom O W; Rosengart T K; Crystal R G (Reprint)
CS CORNELL UNIV, WEILL MED COLL, NEW YORK PRESBYTARIAN HOSP, DIV PULM & CRIT CARE MED, 520 E 70TH ST, NEW YORK, NY 10021 (Reprint); CORNELL UNIV, WEILL MED COLL, NEW YORK PRESBYTARIAN HOSP, DIV PULM & CRIT CARE MED, NEW YORK, NY 10021; NEW YORK HOSP, CORNELL MED CTR, DEPT CARDIOTHORAC SURG, NEW YORK, NY 10021; NEW YORK HOSP, CORNELL MED CTR, DIV CARDIOL, NEW YORK, NY 10021

CYA USA
SO HUMAN GENE THERAPY, (20 MAY 1999) Vol. 10, No. 8, pp. 1331-1348.
Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY 10538.
ISSN: 1043-0342.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 72
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A gene therapy strategy involving direct myocardial administration of an adenovirus (Ad) vector encoding the vascular endothelial **growth factor** 121 **cDNA** (Ad(GV)VEGF121.10) has been shown to be capable of ''biological revascularization'' of ischemic myocardium in an established porcine model [Mack, C.A. (1998). J. Thorac. Cardiovasc. Surg. 115, 168-177]. The present study evaluates the local and systemic safety of this therapy in this porcine ischemia model and in normal mice. Myocardial ischemia was induced in Yorkshire swine with an ameroid constrictor 21 days prior to vector administration. AdGVVEGF121.10 (10(9) or 10(10) PFU), Ad5 wild type (10(9) PFU), AdNull (control vector with no transgene; 10(9) PFU), saline, or no injection (naive) was administered in 10 sites in the ischemic, circumflex distribution of the myocardium. Toxicity was assessed by survival, serial echocardiography, blood analyses, and myocardial and liver histology at 3 and 28 days after vector administration. All pigs survived to sacrifice, except for one animal in the AdGVVEGF121.10 (1010 PFU) group, which died as a result of oversedation. Echocardiograms of **Ad**(GV)VEGF121.10-**treated** pigs demonstrated no differences in pericardial effusion, mitral valve regurgitation, or regional wall motion compared with control pigs. Intramyocardial administration of Ad(GV)VEGF121.10 included only minimal myocardial inflammation and necrosis, and no hepatic inflammation or necrosis. Only a mild elevation of the white blood cell count was encountered on day 3, which was transient and self-limited in the Ad(GV)VEGF121.10 group as compared with the saline-treated animals. As a measure of inadvertent intravascular administration of vector, normal C57/BL6 mice received intravenous Ad(GV)VEGF121.10 (10(4), 10(6), 5 x 10(7), or 10(9) PFU), AdNull (5 x 107 or 109 PFU), or saline. Toxicity was assessed by survival, blood analyses, and organ histology at 3 and 7 days after vector administration. A separate group of C57/BL6 mice received intravenous AdmVEGF164 (Ad vector encoding the murine VEGF164 cDNA), Ad(GV)VEGF121.10, AdNull (10(8) PFU each group), or saline to assess duration of expression and safety of a homologous transgene. All mice survived to sacrifice except for 40% of the mice in the highest (10(9) PFU; a dose more than 10(3)-fold higher by body weight than the efficacious dose in pigs) Ad(GV)VEGF121.10 dose group, which died on days 5-6 after vector administration. The only differences seen in the blood analyses between **treated** and control mice were in the very high **Ad**(GV)VEGF121.10 dose group (109 PFU), which demonstrated an anemia as well as an increase in alkaline phosphatase when compared with all other treatment groups. Hepatic VEGF levels by ELiSA in AdmVEGF164-treated mice did not persist beyond 14 days after vector administration, suggesting that persistent expression of a homologous VEGF gene transferred with an Ad vector is not a significant safety risk. Although this is not a chronic toxicity study, these data demonstrate the safety of direct myocardial administration of Ad(GV)VEGF121.10, and support the potential use of this strategy to treat human myocardial ischemia.

L10 ANSWER 22 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1999:457306 SCISEARCH
GA The Genuine Article (R) Number: 204JA
TI Liposome-mediated NGF gene transfection following neuronal injury: potential therapeutic applications
AU Zou L L; Huang L; Hayes R L; Black C; Qiu Y H; PerezPolo J R; Le W;

Clifton G L; Yang K (Reprint)
CS BAYLOR COLL MED, DEPT NEUROSURG, 1 BAYLOR PLAZA, HOUSTON, TX 77030 (Reprint); BAYLOR COLL MED, DEPT NEUROSURG, HOUSTON, TX 77030; BAYLOR COLL MED, CTR CELL & GENE THERAPY, HOUSTON, TX 77030; BAYLOR COLL MED, DEPT NEUROL, HOUSTON, TX 77030; UNIV PITTSBURGH, DEPT PHARMACOL, PITTSBURGH, PA 15261; UNIV TEXAS, HLTH SCI CTR, DEPT NEUROSURG, HOUSTON, TX; UNIV TEXAS, MED BRANCH, DEPT HUMAN BIOL CHEM & GENET, GALVESTON, TX 77550
CYA USA
SO GENE THERAPY, (JUN 1999) Vol. 6, No. 6, pp. 994-1005.
Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE RG21 6XS,. HAMPSHIRE, ENGLAND.
ISSN: 0969-7128.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 64
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We have systematically investigated the therapeutic potential of cationic liposome-mediated neurotrophic gene transfer for treatment of CNS injury. Following determination of optimal transfection conditions, we examined the effects of dimethylaminoethane-carbamoyl-cholesterol (DC-Chol) liposome-mediated **NGF cDNA** transfection in injured and uninjured primary septo-hippocampal cell cultures and rat brains. In in vitro studies, we detected an increase of NGF mRNA in cultures 1 day after transfection. Subsequent ELISA and PC12 cell biological assays confirmed that cultured cells secreted soluble active NGF into the media from day 2 after gene transfection. Further experiments showed that such NGF gene transfection reduced the loss of choline acetyltransferase (ChAT) activity in cultures following calcium-dependent depolarization injury. In in vivo studies, following intraventricular injections of **NGF cDNA** complexed with DC-Chol liposomes, ELISA detected nine- to 12-fold increases of NGF in rat CSF. Further studies showed that liposome/**NGF cDNA** complexes could attenuate the loss of cholinergic neuronal immunostaining in the rat septum after traumatic brain injury (TBI). Since deficits in cholinergic neurotransmission are a major consequence of TBI, our studies demonstrate for the first time that DC-Chol liposome-mediated NGF gene transfection may have therapeutic potential for **treatment** of **brain** injury.

L10 ANSWER 23 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1999:155236 SCISEARCH
GA The Genuine Article (R) Number: 167DQ
TI Antiangiogenesis treatment for gliomas: Transfer of antisense vascular endothelial growth factor inhibits tumor growth in vivo
AU Im S A; GomezManzano C; Fueyo J; Liu T J; Ke L D; Kim J S; Lee H Y; Steck P A; Kyritsis A P; Yung W K A (Reprint)
CS UNIV TEXAS, MD ANDERSON CANC CTR, DEPT NEUROONCOL, BOX 100, 1515 HOLCOMBE BLVD, HOUSTON, TX 77030 (Reprint); UNIV TEXAS, MD ANDERSON CANC CTR, DEPT NEUROONCOL, HOUSTON, TX 77030; UNIV TEXAS, MD ANDERSON CANC CTR, DEPT HEAD & NECK MED ONCOL, HOUSTON, TX 77030
CYA USA
SO CANCER RESEARCH, (15 FEB 1999) Vol. 59, No. 4, pp. 895-900.
Publisher: AMER ASSOC CANCER RESEARCH, PO BOX 11806, BIRMINGHAM, AL 35202.
ISSN: 0008-5472.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 26
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Presently, there is no effective **treatment** for glioblastoma, the most malignant and common **brain** tumor. Angiogenic factors are potentially optimal targets for therapeutic strategies because they are essential for tumor growth and progression. In this study, we sought a

strategy for efficiently delivering an antisense **cDNA** molecule of the vascular endothelial **growth factor** (VEGF) to glioma cells. The recombinant adenoviral vector Ad5CMV-alpha VEGF carried the coding sequence of wild-type VEGF(165) cDNA in an antisense orientation, Infection of U-87 MG malignant glioma cells with the Ad5CMV-alpha VEGF resulted in reduction of the level of the endogenous VEGF mRNA and drastically decreased the production of the targeted secretory form of the VEGF protein, Treatment of ss. human glioma tumors established in nude mice with intralesional injection of Ad5CMV-alpha VEGF inhibited tumor growth. Taken together, these findings indicate that the efficient down-regulation of the VEGF produced by tumoral cells using antisense strategies has an antitumor effect in vivo. This is the first time that an adenoviral vector is used to transfer antisense VEGF sequence into glioma cells in an animal model, and our results suggest that this system may have clinical and therapeutic utility.

L10 ANSWER 24 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1999:135300 SCISEARCH
GA The Genuine Article (R) Number: 164UH
TI Nerve growth factor expressed in the medial septum following in vivo gene delivery using a recombinant adeno-associated viral vector protects cholinergic neurons from fimbria-fornix lesion-induced degeneration
AU Mandel R J (Reprint); Gage F H; Clevenger D G; Spratt S K; Snyder R O; Leff S E
CS LUND UNIV, WALLENBERG NEUROSCI CTR, NEUROBIOL SECT, SOLVEGATAN 17, S-22362 LUND, SWEDEN (Reprint); CELL GENESYS INC, DEPT PRECLIN BIOL, FOSTER CITY, CA 94404; SALK INST BIOL STUDIES, GENET LAB, LA JOLLA, CA 92037
CYA SWEDEN; USA
SO EXPERIMENTAL NEUROLOGY, (JAN 1999) Vol. 155, No. 1, pp. 59-64.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0014-4886.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Nerve growth factor (NGF) has been shown to support the survival of axotomized medial septal cholinergic neurons after aspirative lesions of the fimbria-fornix (FF). This survival effect has been achieved utilizing intraventricular and intraparenchymal delivery of the NGF protein, While the use of NGF for the **treatment** of the cholinergic deficits present in **Alzheimer**'s disease shows promise based on its efficacy in animal models, concerns about side-effects of intraventricular NGF delivery in humans have been raised. In the present study, NGF was delivered directly to the medial septum via a recombinant adeno-associated viral vector (rAAV) encoding the **cDNA** for human **NGF** prior to a FF lesion in rats. This rAAV-mediated NGF delivery was shown to significantly attenuate the medial septal cholinergic cell loss observed in animals receiving an equivalent injection of a control rAAV vector. (C) 1999 Academic Press.

L10 ANSWER 25 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:163609 CAPLUS
DN 128:226684
TI Human fibroblast growth factor homologous factors and their cDNAs and diagnostic and therapeutic applications
IN Nathans, Jeremy; Smallwood, Philip M.
PA Johns Hopkins University School of Medicine, USA
SO PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9808864	A1	19980305	WO 1997-US15237	19970827
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	US 6020189	A	20000201	US 1996-705245	19960830
	AU 9742406	A1	19980319	AU 1997-42406	19970827
	AU 730403	B2	20010308		
	EP 960117	A1	19991201	EP 1997-940685	19970827
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	BR 9712793	A	19991214	BR 1997-12793	19970827
	NZ 334259	A	20000929	NZ 1997-334259	19970827
	JP 2000517187	T2	20001226	JP 1998-511956	19970827
	US 6635744	B1	20031021	US 2000-490714	20000125
PRAI	US 1996-705245	A	19960830		
	WO 1997-US15237	W	19970827		

AB The invention provides fibroblast growth factor homologous factor (FHF) polypeptides and nucleic acid mols. that encode them. Thus, cDNA sequences and their deduced amino acid sequences are provided for 4 human-derived FHFs. The amino acid sequence of FHF-1 is 27% identical to that of fibroblast growth factor-9, and the amino acid sequences of FHFs 1-4 are 58-70% identical to each other, thus defining a new branch of the FGF family. The genes for FHF-1, -2, -3, and -4 are located on human chromosomes 3, X, 17, and 13, resp. Each FHF is expressed in the brain, and FHF-1, FHF-2 and FHF-3 are expressed in the eye, FHF-1 and FHF-4 are expressed in the testis, and FHF-2 is expressed in the heart. FHFs are involved in regulating the growth, survival, and differentiation of cells in the central nervous system, as well as cells in peripheral nervous tissues. Also included in the invention are diagnostic and therapeutic methods using FHF polypeptides and nucleic acids. Regions of conserved amino acid sequence can be used to design oligonucleotide primers/probes, and antibodies can be generated for diagnostic and therapeutic applications.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 26 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 1998:368339 SCISEARCH
GA The Genuine Article (R) Number: ZM121
TI Responses of young and aged rat CNS to partial cholinergic immunolesions and NGF treatment
AU Wortwein G; Yu J; ToliverKinsky T; PerezPolo J R (Reprint)
CS UNIV TEXAS, MED BRANCH, DEPT HUMAN BIOL CHEM & GENET, GALVESTON, TX 77555 (Reprint); UNIV TEXAS, MED BRANCH, DEPT HUMAN BIOL CHEM & GENET, GALVESTON, TX 77555; RIGSHOSP, LAB NEUROPSYCHIAT, DK-2100 COPENHAGEN, DENMARK
CYA USA; DENMARK
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1 MAY 1998) Vol. 52, No. 3, pp. 322-333.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0360-4012.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 76
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The cholinergic neurons of the basal forebrain (CNBF) are the major source of cholinergic innervation of the cortex and hippocampus, In Alzheimer's disease and aged brain, there are severe losses of cholinergic neurons in the nucleus basalis of Meynert, leading to a reduction of cortical cholinergic activity which correlates with the severity of cognitive deficits. While there is evidence that aged **central nervous system** (CNS) displays impaired stress response signaling, pharmacologic **treatments** with neurotrophic factors appear to ameliorate these age-associated cholinergic deficits. To mimic these cholinergic deficits in experimental animals and study the acute effects of nerve growth factor (NGF), we induced a partial lesion of CBFNs by the intracerebroventricular (icv) injection of the cholinergic immunotoxin 192IgG-saporin, in groups of 3- and 30-month-old rats. The lesion was followed 14 days later by icy administration of NGF, known to restore partial immunolesion-induced cholinergic deficits in rat CNS, and all rats were killed 2 days after the NGF treatment, Here we report the effects of partial immunolesions on the levels of choline acetyltransferase (ChAT) activity and NGF receptor mRNA levels in the basal forebrain of 3- and 30-month-old rats. Because of their presence in the promoters of the NGF, **NGF** receptors, and ChAT genes, we also measured **DNA**-binding activity of the transcription factors NFB and AP-1 in the cortex and hippocampus, We discuss these findings in the context of endogenous NGF-mediated signal transduction mechanisms and conclude that we have evidence for age-associated decreases in endogenous NGF responses to partial deafferentation of the basal forebrain cholinergic projections. (C) 1998 Wiley-Liss, Inc.

L10 ANSWER 27 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:696876 CAPLUS
DN 128:10673
TI A neuronal cell activity regulated pentraxin as a neuronal cell growth factor and a cDNA encoding it
IN Worley, Paul; Tsui, Cynthia
PA Johns Hopkins University School of Medicine, USA
SO PCT Int. Appl., 104 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9739133	A1	19971023	WO 1997-US5694	19970408
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	US 5767252	A	19980616	US 1996-631607	19960408
	AU 9726084	A1	19971107	AU 1997-26084	19970408
	US 6436673	B1	20020820	US 1998-98358	19980616
	US 2003022313	A1	20030130	US 2002-224951	20020820
PRAI	US 1996-631607	A	19960408		
	WO 1997-US5694	W	19970408		
	US 1998-98358	A1	19980616		

AB A neuronal activity regulated pentraxin (Narp) that modulates neuronal cell growth of rat is characterized and a cDNA encoding it is cloned. Narp is useful for induction of dendritic neurite outgrowth as well as promotion of neuronal migration and can be used in the treatment of neurol. diseases. A cDNA was obtained from a subtracted cDNA library of rat hippocampus prepd. 4 h after electroconvulsive seizure. The mRNA for the protein is present in sensory ganglia and limbic structures and shows

development regulation. The protein is manufd. as a precursor with a signal peptide and is secreted. The protein shows quant. binding to a polysaccharide (agar) in the presence of calcium.

L10 ANSWER 28 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 97:662100 SCISEARCH
GA The Genuine Article (R) Number: XU081
TI Regulation of nerve growth factor secretion and mRNA expression by bacterial lipopolysaccharide in primary cultures of rat astrocytes
AU GalveRoperh I; Malpartida J M; Haro A; Brachet P; DiazLaviada I (Reprint)
CS UNIV ALCALA DE HENARES, DEPT BIOCHEM, FAC MED, ALCALA DE HENARES 28871, MADRID, SPAIN (Reprint); UNIV ALCALA DE HENARES, DEPT BIOCHEM, FAC MED, ALCALA DE HENARES 28871, MADRID, SPAIN; UNIV ALCALA DE HENARES, DEPT BIOCHEM & MOL BIOL, FAC MED, ALCALA DE HENARES 28871, MADRID, SPAIN; UNIV COMPLUTENSE, DEPT BIOCHEM & MOL BIOL, FAC BIOL, E-28040 MADRID, SPAIN; CTR HOSP REG UNIV, INSERM U298, ANGERS, FRANCE
CYA SPAIN; FRANCE
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1 SEP 1997) Vol. 49, No. 5, pp. 569-575.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0360-4012.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 35
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The present work was undertaken to study the effect of bacterial lipopolysaccharide (LPS), a potent activator of the host inflammatory response, on the synthesis of nerve growth factor (NGF) by newborn rat **brain** astrocytes. **Treatment** of primary rat astroglial cells cultured in chemically defined medium with LPS resulted in a dose-dependent accumulation of NGF mRNA, and an increased release of NGF protein in the cell medium. NGF mRNA levels were maximal after 24 hr of stimulation (8-fold increase), whereas extracellular NGF peaked after 72 hours of treatment (17-fold increase). This dramatic increase of extracellular NGF was abrogated if cells were treated with actinomycin D or cycloheximide, a fact which implies that the accumulation of extracellular **NGF** by UPS-treated cells requires **DNA** transcription and RNA translation. Stimulation of **NGF** synthesis and secretion was: (i) unaffected by treatment with the protein kinase C inhibitor bisindolylmaleimide, and (ii) prevented by forskolin and 3-isobutyl-1-methylxanthine, two agents which increase cAMP levels. Inhibition of LPS effect was also obtained with apigenin, a proposed inhibitor of the mitogen-activated protein kinase pathway. Results thus show that LPS stimulates NGF synthesis by astroglial cells through a mechanism that is independent of protein kinase C (PKC), antagonized by cAMP-elevating agents, and probably mediated by the mitogen-activated protein kinase cascade. The data raise the possibility that LPS exerts stimulatory effects on NGF synthesis that are independent of those elicited by astrocyte-derived inflammatory lymphokines such as IL-1 beta, TNF alpha or TGF beta 1. (C) 1997 Wiley-Liss, Inc.

L10 ANSWER 29 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:47273 CAPLUS
DN 126:99589
TI Neurogenesis in neonatal rat brain is regulated by peripheral injection of basic fibroblast growth factor (bFGF)
AU Tao, Y.; Black, I. B.; Dicicco-Bloom, E.
CS Robert Wood Johnson Medical School, UMDNJ, Piscataway, NJ, 08854, USA
SO Journal of Comparative Neurology (1996), 376(4), 653-663
CODEN: JCNEAM; ISSN: 0021-9967
PB Wiley-Liss
DT Journal

LA English
AB Many major diseases of human brain involve deficiencies of select neuronal populations. As one approach to repair, the authors examd. regulation of neurogenesis directly in vivo, employing postnatal day 1 (P1) cerebellar cortex, which is composed primarily of granule neurons and dividing precursors. The authors focused on basic fibroblast growth factor (bFGF), which stimulates precursor mitosis in culture and which is highly expressed in cerebellum during neurogenesis. S.c. injection of bFGF increased [3]thymidine ([3H]dT) incorporation, a marker for DNA synthesis, by 50% in whole cerebellar homogenates, suggesting that peripherally administered factor altered ongoing neural proliferation. Further, assay of isolated granule precursors revealed a 4-fold increase in [3H]dT incorporation following in vivo bFGF treatment, indicating that granule neuroblasts were the major bFGF-responsive population. Morphol. anal. indicated that twice as many granule precursors were in S-phase of the mitotic cycle after peripheral bFGF. To det. whether other neurogenetic populations respond to peripheral bFGF, the authors examd. addnl. brain regions in vivo. BFGF stimulated DNA synthesis by 68% in hippocampus, and by > 250% in pontine subventricular zone (SVZ). In contrast, incorporation was not altered in basal pons or cerebral cortex, regions in which neurogenesis has already ceased. To define potential direct actions of peripherally administered factor, 125I-bFGF was used to study distribution. Intact 18 kDa 125I-bFGF was recovered from brain following peripheral injection, suggesting that the factor acted directly to stimulate mitosis in dividing neuroblasts. The stimulation of neuronal proliferation by exogenous bFGF suggests that the factor normally regulates neurogenesis, and provides new therapeutic approaches to promote functional recovery from nervous system diseases.
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 30 OF 38 MEDLINE on STN DUPLICATE 3
AN 96290680 MEDLINE
DN 96290680 PubMed ID: 8730841
TI Liposome-mediated BDNF cDNA transfer in intact and injured rat brain.
AU Iwamoto Y; Yang K; Clifton G L; Hayes R L
CS Department of Neurosurgery, University of Texas Houston Health Science Center, Houston 77030, USA.
NC PO1 NS31998 (NINDS)
RO1 NS21458 (NINDS)
SO NEUROREPORT, (1996 Jan 31) 7 (2) 609-12.
Journal code: 9100935. ISSN: 0959-4965.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199611
ED Entered STN: 19961219
Last Updated on STN: 19961219
Entered Medline: 19961125
AB We examined the temporal profile of the expression of brain-derived neurotrophic factor (BDNF) cDNA containing a viral promotor following the injection of liposome cDNA complexes into the intact and traumatically injured rat brain. In situ hybridization and PCR confirmed the presence of injected BDNF cDNA for at least 6 days after injection. A similar profile of BDNF cDNA was observed when it was injected following cortical impact injury. mRNA was also localized around the injection areas. These results suggest that liposome-mediated delivery of **neurotrophin cDNA** may be a practical gene transfer method for **treating** traumatic **brain** injury.

L10 ANSWER 31 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:663654 CAPLUS
DN 126:128522

TI Cloning of a serine proteinase inhibitor from bovine brain: expression in the brain and characterization of its target proteinases
AU Nakaya, Naoki; Nishibori, Masahiro; Kawabata, Masahiro; Saeki, Kiyomi
CS Department of Pharmacology, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama, 700, Japan
SO Molecular Brain Research (1996), 42(2), 293-300
CODEN: MBREE4; ISSN: 0169-328X
PB Elsevier
DT Journal
LA English
AB A cDNA encoding the serine proteinase inhibitor (serpin), B-43, was cloned from the cDNA library of the bovine brain. It encoded 378 amino acids, and the MW of the protein was estd. to be 42.6 kDa, which is consistent with that of the native B-43 purified from the bovine brain. The homol. search revealed that B-43 belongs to the ovalbumin branch of the serpin superfamily. Among them, B-43 was most homologous to human placental thrombin inhibitor (PI-6) and its murine counterpart, with the amino acid identity of 76 and 71, resp. Northern blot anal. showed that the size of the transcript was 1.4 kb, and that the expression of B-43 in the bovine brain varied depending on the brain regions, i.e. a lower level of expression was obsd. in the cerebral cortex and the hippocampus compared to the level of expression that was obsd. in the medulla oblongata. [35S]-labeled B-43 protein was synthesized in vitro by using a rabbit reticulocyte lysate system, which formed complexes with proteinases such as thrombin, trypsin, .alpha.-chymotrypsin, and 7S nerve growth factor (NGF), but not with urokinase or plasmin. These results, together with the immunohistochem. localization of B-43 in astrocytes and in some neurons which was obsd. in the previous study suggest that B-43 may be involved in the regulation of serine proteinases present in the brain or extravasated from the blood.
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 32 OF 38 MEDLINE on STN DUPLICATE 4
AN 96075561 MEDLINE
DN 96075561 PubMed ID: 7586219
TI VEGF165 expressed by a replication-deficient recombinant adenovirus vector induces angiogenesis in vivo.
AU Muhlhauser J; Merrill M J; Pili R; Maeda H; Bacic M; Bewig B; Passaniti A; Edwards N A; Crystal R G; Capogrossi M C
CS Pulmonary Branch, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD, USA.
SO CIRCULATION RESEARCH, (1995 Dec) 77 (6) 1077-86.
Journal code: 0047103. ISSN: 0009-7330.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199512
ED Entered STN: 19960124
Last Updated on STN: 19960124
Entered Medline: 19951226
AB To evaluate the concept that localized delivery of angiogenic factors via virus-mediated gene transfer may be useful in the **treatment** of ischemic disorders, the replication-deficient adenovirus (**Ad**) vector AdCMV.VEGF165 (where CMV is cytomegalovirus and VEGF is vascular endothelial **growth factor**) containing the **cDNA** for human VEGF165, a secreted endothelial cell-specific angiogenic growth factor, was constructed. Human umbilical vein endothelial cells (HUVECs) and rat aorta smooth muscle cells (RASMCs) infected with AdCMV.VEGF165 (5 and 20 plaque-forming units [pfu] per cell) demonstrated VEGF mRNA expression and protein secretion into the supernatant. Furthermore, the conditioned medium from these cells enhanced vascular permeability in vivo. In contrast, neither VEGF mRNA nor secreted protein was found in

uninfected HUVECs or RASMCs or in cells infected with the control vector AdCMV.beta gal (where beta gal is beta-galactosidase). Assessment of starved HUVECs at 14 days demonstrated sixfold more cells for AdCMV.VEGF165-infected HUVECs (20 pfu per cell) than for either infected or uninfected control cells. RASMC proliferation was unaffected by infection with AdCMV.VEGF165. When plated in 2% serum on dishes precoated with reconstituted basement membrane (Matrigel), HUVECs infected with AdCMV.VEGF165 (20 pfu per cell) differentiated into capillary-like structures. Under similar conditions, both uninfected HUVECs and HUVECs infected with AdCMV.beta gal did not differentiate. To evaluate the ability of AdCMV.VEGF165 to function in vivo, either AdCMV. VEGF165 or AdCMV.beta gal (2 x 10(10) pfu) was resuspended in 0.5 mL Matrigel and injected subcutaneously into mice. Immunohistochemical staining demonstrated VEGF in the tissues surrounding the Matrigel plugs containing AdCMV.VEGF165 up to 3 weeks after injection, whereas no VEGF was found in the control plugs with AdCMV.beta gal. Two weeks after injection, there was histological evidence of neovascularization in the tissues surrounding the Matrigel containing AdCMV.VEGF165, whereas no significant angiogenesis was observed in response to AdCMV.beta gal. Furthermore, the Matrigel plugs with AdCMV.VEGF165 demonstrated hemoglobin content fourfold higher than the plugs with AdCMV.beta gal. Together, these in vitro and in vivo studies are consistent with the concept that Ad vectors may provide a useful strategy for efficient local delivery of VEGF165 in the treatment of ischemic diseases.

L10 ANSWER 33 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 95:561977 SCISEARCH
GA The Genuine Article (R) Number: RP839
TI NEUROCHEMICAL EFFECTS OF A 20 KHZ MAGNETIC-FIELD ON THE CENTRAL-NERVOUS-SYSTEM IN PRENATALLY EXPOSED MICE
AU DIMBERG Y (Reprint)
CS SWEDISH UNIV AGR SCI, DEPT RADIOECOL, BOX 7030, S-75007 UPPSALA, SWEDEN (Reprint)
CYA SWEDEN
SO BIOELECTROMAGNETICS, (1995) Vol. 16, No. 4, pp. 263-267.
ISSN: 0197-8462.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB C57/B1 mice were exposed during pregnancy (gestation days 0-19) to a 20 kHz magnetic field (MF). The asymmetric sawtooth-waveform magnetic field in the exposed racks had a flux density of 15 mu T (peak to peak). After 19 days, the exposure was terminated, and the mice were housed individually under normal laboratory conditions. On postnatal day (PD) 1, PD21, and PD308, various neurochemical markers in the brains of the offspring were investigated and the brains weighed. No significant difference was found in the whole brain weight at PD1 or PD21 between exposed offspring and control animals. However, on PD308, a significant **decrease** in weight of the whole **brain** was detected in exposed animals. No significant differences were found in the weight of cortex, hippocampus, septum, or cerebellum on any of the sampling occasions, nor were any significant differences detected in protein-, **DNA**-level, nerve **growth factor** (**NGF**), acetylcholine esterase- (AChE), or 2',3'-cyclic nucleotide 3'-phosphodiesterase- (CNP; marker for oligodendrocytes) activities on PD21 in cerebellum. Cortex showed a more complex pattern of response to MF: MF treatment resulted in a decrease in DNA level and increases in the activities of CNP, AChE, and **NGF** protein. On PD308, the amount of **DNA** was significantly reduced in MF-treated cerebellum and CNP activity was still enhanced in MF-treated cortex compared to controls. Most of the effects of MF treatment during the embryonic period were similar to those induced by ionizing radiation but much weaker. However,

the duration of the exposure required to elucidate the response of different markers to MF seems to be greater and effects appear later during development compared to responses to ionizing radiation. (C) 1995 Wiley-Liss, Inc.

L10 ANSWER 34 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 94:674771 SCISEARCH
GA The Genuine Article (R) Number: PM494
TI BRAIN GROWTH-RETARDATION DUE TO THE EXPRESSION OF HUMAN INSULIN-LIKE GROWTH-FACTOR BINDING PROTEIN-1 IN TRANSGENIC MICE - AN IN-VIVO MODEL FOR THE ANALYSIS OF IGF FUNCTION IN THE BRAIN
AU DERCOLE A J (Reprint); DAI Z H; XING Y Z; BONEY C; WILKIE M B; LAUDER J M; HAN V K M; CLEMMONS D R
CS UNIV N CAROLINA, DEPT PEDIAT, CB 7220, CHAPEL HILL, NC, 27599 (Reprint); UNIV N CAROLINA, DEPT MED, CHAPEL HILL, NC, 27599; UNIV N CAROLINA, DEPT CELL BIOL & ANAT, CHAPEL HILL, NC, 27599; LAWSON RES INST, LONDON N6A 4V2, ON, CANADA; UNIV WESTERN ONTARIO, ST JOSEPHS HLTH CTR, LONDON N6A 4V2, ON, CANADA
CYA USA; CANADA
SO DEVELOPMENTAL BRAIN RESEARCH, (14 OCT 1994) Vol. 82, No. 1-2, pp. 213-222. ISSN: 0165-3806.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 58
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Three lines of transgenic (Tg) mice carrying a fusion gene linking the mouse metallothionein-I promoter to a **cDNA** encoding human insulin-like **growth factor** binding protein-1 (hIGFBP-1) were found to express the transgene in brain. As judged by comparing Tg brain weights to those of non-transgenic littermates, adult hemizygotic Tg mice of each line exhibited brain growth retardation (16.2%, 14.4% and 8.1% reductions in weight, respectively in each line). In two lines, total **brain** DNA and protein content were **decreased**. Further analysis indicated that the **brain** growth retardation was manifested in the second week of postnatal life. Given that the insulin-like growth factors (IGFs) stimulate cell proliferation and/or survival in neural cultures and that hIGFBP-1, when present in a molar excess, **inhibits** IGF interactions with their cell surface receptors, the **brain** growth retardation in hIGFBP-1 Tg mice likely results from hIGFBP-1 inhibition of IGF-stimulated growth-promoting actions. These hIGFBP-1 Tg mice should prove useful in defining IGF actions during postnatal brain maturation.

L10 ANSWER 35 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 93:719211 SCISEARCH
GA The Genuine Article (R) Number: MK424
TI TEMPORAL ANALYSIS OF EVENTS ASSOCIATED WITH PROGRAMMED CELL-DEATH (APOPTOSIS) OF SYMPATHETIC NEURONS DEPRIVED OF NERVE GROWTH-FACTOR
AU DECKWERTH T L; JOHNSON E M (Reprint)
CS WASHINGTON UNIV, SCH MED, DEPT MOLEC BIOL & PHARMACOL, 660 S EUCLID AVE, BOX 8103, ST LOUIS, MO, 63110
CYA USA
SO JOURNAL OF CELL BIOLOGY, (DEC 1993) Vol. 123, No. 5, pp. 1207-1222. ISSN: 0021-9525.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 85
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The time course of molecular events that accompany degeneration and death after nerve growth factor (NGF) deprivation and neuroprotection by NGF and other agents was examined in cultures of NGF-dependent neonatal rat sympathetic neurons and compared to death by apoptosis. Within 12 h

after onset of NGF deprivation, glucose uptake, protein synthesis, and RNA synthesis fell precipitously followed by a moderate decrease of mitochondrial function. The molecular mechanisms underlying the NGF deprivation-induced decrease of protein synthesis and neuronal death were compared and found to be different, demonstrating that this decrease of protein synthesis is insufficient to cause death subsequently. After these early changes and during the onset of **neuronal atrophy**, **inhibition** of protein synthesis ceased to halt neuronal degeneration while readdition of NGF or a cAMP analogue remained neuroprotective for 6 h. This suggests a model in which a putative killer protein reaches lethal levels several hours before the neurons cease to respond to readdition of NGF with survival and become committed to die. Preceding loss of viability by 5 h and concurrent with commitment to die, the neuronal DNA fragmented into oligonucleosomes. The temporal and pharmacological characteristics of DNA fragmentation is consistent with DNA fragmentation being part of the mechanism that commits the neuron to die. The antimitotic and neurotoxin cytosine arabinoside induced **DNA** fragmentation in the presence of **NGF**, supporting previous evidence that it mimicked NGF deprivation-induced death closely. Thus trophic factor deprivation-induced death occurs by apoptosis and is an example of programmed cell death.

L10 ANSWER 36 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 92:292447 SCISEARCH
GA The Genuine Article (R) Number: HR339
TI REGULATION OF INSULIN-LIKE GROWTH FACTOR-I PRODUCTION IN RAT C6 GLIOMA-CELLS - POSSIBLE ROLE AS AN AUTOCRINE PARACRINE GROWTH-FACTOR
AU LOWE W L (Reprint); MEYER T; KARPEN C W; LORENTZEN L R
CS VET ADM MED CTR, DEPT INTERNAL MED, ROOM 3E-17, IOWA CITY, IA, 52246 (Reprint); UNIV IOWA, COLL MED, DEPT INTERNAL MED, IOWA CITY, IA, 52242
CYA USA
SO ENDOCRINOLOGY, (MAY 1992) Vol. 130, No. 5, pp. 2683-2691. ISSN: 0013-7227.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 49
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The growth of rat glioma C6 cells, which provide an in vitro model of glial cells, is inhibited by retinoic acid and glucocorticoids, two agents which are important in **brain** differentiation and growth. To determine whether the growth-**inhibitory** effects of these agents are mediated by alterations in insulin-like growth factor I (IGF-1) production, the effects of retinoic acid and dexamethasone on IGF-I production and messenger RNA levels in C6 cells were investigated. IGF-I mRNA levels were determined using a solution hybridization/RNase protection assay. Treatment of C6 cells with dexamethasone or retinoic acid decreased IGF-I mRNA levels in a time-dependent fashion. The time course of the effect of the two agents differed, with the peak effect of dexamethasone between 6 and 12 h and the peak effect of retinoic acid at 27 h. In dose-response studies, IGF-I mRNA levels decreased to 27% of control levels (cells maintained in serum-free media) after treatment with 5 ng/ml dexamethasone, while half-maximal inhibition was achieved with approximately 0.5 ng/ml (1.4 nm) dexamethasone. Treatment with 10-mu-m retinoic acid decreased IGF-I mRNA levels to 24% of control levels with half-maximal inhibition occurring with approximately 0.5-mu-m retinoic acid. Cycloheximide prevented the inhibitory effect of these agents on IGF-I mRNA levels, suggesting that their effect is at least partly dependent upon protein synthesis. Immunoreactive IGF-I levels in media conditioned for 48 h by cells treated with dexamethasone or retinoic acid decreased to 32% and 42% of control levels, respectively. Treatment of C6 cells with retinoic acid or dexamethasone decreased thymidine incorporation into DNA. Treatment of cells with IGF-I alone had no effect on thymidine incorporation into DNA, but addition of 10 or 50 ng/ml IGF-I

to dexamethasone-treated cells stimulated a small, but significant (P < 0.01), increase in thymidine incorporation into DNA. IGF-I was not, however, able to reverse the inhibitory effect of retinoic acid. Finally, treatment of cells with 150 ng/ml of IGF binding protein 1 significantly decreased (P < 0.01) thymidine incorporation into DNA by 17% as compared to incorporation into control cells maintained in serum- free media. While the role of IGF-I in retinoic acid-induced alterations in C6 cell growth is unclear, the ability of IGF-I to partially reverse the dexamethasone-induced inhibition of thymidine incorporation into DNA suggests that the inhibitory effect of dexamethasone on C6 cell growth may be mediated in part by alterations in IGF-I production and that, taken together with the effect of IGF binding protein 1 on thymidine incorporation into **DNA**, IGF-I may be an autocrine/paracrine **growth factor** in these cells.

L10 ANSWER 37 OF 38 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 92:554202 SCISEARCH
GA The Genuine Article (R) Number: JN576
TI EXPRESSION OF THE EPIDERMAL GROWTH-FACTOR RECEPTOR GENE IN HUMAN BRAIN METASTASES
AU TORP S H (Reprint); HELSETH E; RYAN L; STOLAN S; DALEN A; UNSGAARD G
CS MED TECH CTR, INST CANC RES, N-7005 TRONDHEIM, NORWAY (Reprint); MED TECH CTR, DEPT NEUROSURG, N-7005 TRONDHEIM, NORWAY; MED TECH CTR, UNIGEN, N-7005 TRONDHEIM, NORWAY; MED TECH CTR, DEPT MICROBIOL, N-7005 TRONDHEIM, NORWAY
CYA NORWAY
SO APMIS, (AUG 1992) Vol. 100, No. 8, pp. 713-719.
ISSN: 0903-4641.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 43
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Biopsy specimens of human brain metastases were examined for amplification and expression of the proto-oncogene c-erbB1 (located on chromosome 7) encoding the epidermal **growth factor** receptor (EGFR). Moreover, the tumour **DNA** was also examined for amplification of other cancer-related genes on this chromosome: the proto-oncogene c-met, the gene for platelet-derived growth factor A-chain, and the gene for plasminogen activator **inhibitor** type 1. All 18 **brain** metastases demonstrated positive binding of biotinylated EGF on cryosections. Three out of 18 metastases had amplification of the EGFR gene; the other chromosome-7 genes tested were not amplified. Thus, an increased EGFR gene expression seems to be a general finding in a wide range of carcinomas metastatic to the brain, whereas we found only occasional selective EGFR gene amplifications in single cases.

L10 ANSWER 38 OF 38 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1991:466201 CAPLUS
DN 115:66201
TI Cloning and expression of neurotrophin-3 (NT-3) cDNA and use of NT-3 for diagnosis and treatment of neurologic disorders
IN Hohn, Andreas; Leibrock, Joachim; Bailey, Karen; Barde, Yves Alain; Thoenen, Hans F. E.; Maisonpierre, Peter C.; Furth, Mark E.; Lindsay, Ronald M.; Yancopoulos, George
PA Max-Planck-Gesellschaft zur Foerderung der Wissenschaften e.V., Germany; Regeneron Pharmaceuticals, Inc.
SO PCT Int. Appl., 149 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 9103569 A1 19910321 WO 1990-US4916 19900829
W: AU, BB, BG, BR, CA, DK, ES, FI, HU, KR, LK, MC, MG, MW, NO, RO, SD, SU
RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, IT, LU, ML, MR, NL, SE, SN, TD, TG
US 5180820 A 19930119 US 1989-400591 19890830
IL 95511 A1 20001031 IL 1990-95511 19900828
CA 2040437 AA 19910301 CA 1990-2040437 19900829
CA 2040437 C 20021022
AU 9064049 A1 19910408 AU 1990-64049 19900829
AU 643705 B2 19931125
EP 441947 A1 19910821 EP 1990-913954 19900829
EP 441947 B1 19970514
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE
AT 153074 E 19970515 AT 1990-913954 19900829
ES 2100891 T3 19970701 ES 1990-913954 19900829
RU 2128226 C1 19990327 RU 1990-4895662 19900829
SG 70560 A1 20000222 SG 1996-7705 19900829
CN 1052141 A 19910612 CN 1990-108379 19900830
ZA 9006926 A 19910626 ZA 1990-6926 19900830
ZA 9006927 A 19910626 ZA 1990-6927 19900830
DD 299196 A5 19920402 DD 1990-343740 19900830
DD 299197 A5 19920402 DD 1990-343741 19900830
JP 05161493 A2 19930629 JP 1990-229505 19900830
JP 05328974 A2 19931214 JP 1990-229504 19900830
JP 2783450 B2 19980806
SK 279660 B6 19990211 SK 1990-4230 19900830
CZ 285649 B6 19991013 CZ 1990-4229 19900830
CZ 286015 B6 19991215 CZ 1990-4230 19900830
NO 9101688 A 19910531 NO 1991-1688 19910429
US 5453361 A 19950926 US 1992-823117 19920121
LV 10792 B 19951220 LV 1993-1135 19931006
LT 4011 B 19960826 LT 1993-1546 19931207
LT 4063 B 19961125 LT 1994-1818 19940128
PRAI US 1989-400591 A 19890830
US 1990-490004 A 19900307
US 1990-570189 A 19900820
US 1990-570657 A 19900820
WO 1990-US4916 A 19900829

AB Genomic DNA encoding NT-3, a novel member of the nerve growth factor (NGF)/brain-derived neurotrophic factor (BDNF) gene family, are cloned from mice, rats, and human and characterized. Their amino acid sequences are deduced and the biol. activities of their expression products detd. Oligonucleotides corresponding to regions highly conserved between NGF and BDNF were prepd. and used in the polymerase chain reaction to prep. probes for cloning the NT-3 gene from mammals. The genes encoding mature NT-3 from rats and human were 92% homologous and both supported the fiber outgrowth of chick embryo dorsal root ganglia or nodose ganglia in explant culture. The expression profiles of the genes for NT-3, NGF, and BDNF at various developmental and growth stages were also examd. in the rat central nervous system.